Sveriges lantbruksuniversitet
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and Animal Science
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Degree Project 30 credits within the Veterinary Medicine Programme

ISSN 1652-8697
Examensarbete 2014:54

Uppsala
2014

Complications to strangles in horses presented at referral hospitals
Komplikationer till kvarka hos hästar inkommande till remitterande hästsjukhus

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Degree Project in Veterinary Medicine

***Credits**: 30 hec*
***Level**: Second cycle, A2E*
***Course code**: EX0736*

***Place of publication**: Uppsala*
***Year of publication**: 2014*
***Number of part of series**: Examensarbete 2014:54*
***ISSN**: 1652-8697*
***Online publication**: http://stud.epsilon.slu.se*

***Key words**: Stangles, equine, horses, complication Streptococcus equi subsp. equi*
***Nyckelord**: Kvarka, häst, komplikationer, Streptococcus equi subsp. equi*

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SAMMANFATTNING

Kvarka är en smittsam luftvägssjukdom som drabbar hästar. Kvarka orsakas av bakterien *Streptococcus equi* supsp. *equi*. Den klassiska symptombilden är feber, näsflöde (24-48 timmar efter feber) först tunt och klart sekret sedan mer tjockt samt svullna lymfknutor och svårigheter att svälja. De svullna lymfknutorna kan bilda abscesser som sedan kan spricka upp med varutträde. Det är de unga hästarna som visar kraftigast symptom och de äldre hästarna mildare. Bakterien sprids både direkt och indirekt och utsöndring av bakterier börjar 24-48 timmar efter första febertoppen. Inkubationstiden är 3-14 dagar och det är när utsöndringen av bakterien börjar som provtagning kan ske. Nässvabbsprov, nässköljsprov och provtagning av purulent exsudat och analys via odling och PCR används. Symtomlösa smittbärare som intermittent utsöndrar bakterier från luftsäckarna är en trolig smittspridare.

Kvarka kan efter infektionens utbrott leda till olika följdsjukdomar. Dessa kan vara allvarliga och leda till stora djurlidanden. De mest omskrivna komplikationerna är lunginflammation, kastad kvarka, anasarka (purpura hemorrhagica) och myosit.

I denna retrospektiva studie ingick 69 hästar remitterade till hästsjukhus på Universitetsdjursjukhuset SLU samt Regionhästsjukhuset Helsingborg. Av dessa drabbades fyra hästar av ovannämda komplikationer. De vanligaste symptomen var feber och dysfagi. De vanligaste komplikationerna var svullna retropharyngeallymfknutor och purulent exsudat i luftsäckarna. Det fanns en korrelation mellan svullen pharynx och dysfagi och feber. Det fanns även en korrelation mellan svullna retropharyngeallymfknutor, näsflöde och dyspné.

Fem av hästarna avlivades varav tre på grund av ekonomiska skäl, en i djurskyddssynpunkt och en självdog på kliniken. En sjätte häst dog hemma, uppgifter om orsak saknas. 32 hästägare kontaktades per telefon för uppföljande kontakt om hur hästens sjukdom utvecklats efter hemgång. Av dessa hade 27 hästar tillfrisknat och fyra hade avlivats.

SUMMARY

Strangles is a highly contagious disease in horses. Strangles is caused by the bacteria *Streptococcus equi* subsp. *equi.* Common clinical signs are fever, nasal discharge (24-48 h after onset of fever), swollen lymph nodes and problems swallowing. The swollen lymph nodes may form abscesses that may erupt. Younger horses suffer from more severe clinical signs than older horses. The bacterium is spread within 24-48 hours after the first onset of fever and is transmitted either direct or indirect. The incubation time is 3-14 days and when transmission of the bacteria begins samples can be taken. The diagnosis of strangles is based on clinical symptoms and laboratory findings like culture, PCR and/or serology. But culture of nasal swabs, nasal washes and aspirated pus from abscesses remains the gold standard. Asymptomatic carriers that intermittently shed the bacteria are a source of transmission. Strangles may lead to several complications. These are severe and cause loss in animal welfare. Pneumonia, bastard strangles, purpura hemorrhagica and myositis are the most mentioned common complicatiations in the literature.

In this retrospective study included 69 horses referred to two equine hospitals. Four of them developed the above-mentioned complications. The most common presenting clinical signs were fever and dysphagia. The most common clomplications were swollen retoropharyngal lymph nodes and purulent discharge in the guttural pouch. There was a correlation between swollen pharynx and dysphagia and fever. There was also a correlation between swollen retropharyngal lymphnodes and nasal discharge and dyspnea.

Five of the horses were euthanized, of which three were because of economic reasons, one because of animal welfare and one died by natural causes at the hospital. A 6th horse died at home and no cause of death was recorded.

32 owners were contacted by telephone for a follow-up. 27 horses recovered fully and four horses were euthanized.

Complication ratesand case fatality in horses referred for care of strangles in Sweden appeared similar to what was reported in field cases, with the exception that no diagnosis of myositis was observed in these horses.

CONTENTS

INTRODUCTION

Strangles is a highly contagious disease caused by *Streptococcus equi* subsp. *equi*. The aim of this study was to assess the type and extent of complications in horses with strangles that are referred to equine hospitals. American studies report complication rates but are largely from the field and case reports.

This study aims to describe the situation in Sweden and focused only on horses referred or admitted to referral centers for hospital care.

The complication caused by strangles may be severe and lead to animal welfare issues and may be a reason for euthanasia. It is therefore important to identify the incidence of complications from strangles.

MATERIALS AND METHODS

A retrospective study. A review of journals from the system Trofast from the University Equine Clinic and the system VetVision at the Equine Hospital in Helsingborg. Journals from 2003-01-01 to 2013-01-01 were analysed. Since strangles is a notifiable disease the recovery of appropriate journals was straight forward through use of the search word was ”Strangles” (sv “Kvarka”).

The journals were reviewed and sorted in excel according to clinical signs present when the horses arrived to the hospital and the complications caused by the bacteria during the period of hospitalization. Complications were confirmed by endoscopy and appropriate diagnostic samples. The clinical signs were sorted into 15 different groups. The complications were sorted into 19 groups. Many horses had several clinical signs and/or complications.

The clinical signs were divided into 15 groups. Dysphagia was seperated from inappetence. Dysphagia includes problem with food intake, swallowing and chewing whereas inappetence describes a horse that may chew and swallow but is not interested in its food. Dyspnea and respiratory distress formed another group of horses including irregular respiratory rate and lung sounds, and was seperated from choughing. The horses suffering from nasal discharge was seperated in another group since nasal dischage is well known as the most characteristic clinical sign of strangles. The group no clinical signs regarded horses in stables infected with strangles but did not have any clinical signs on its own. Horses suffering from colic had decreased intestinal sounds, findings by rectal palpation and/or abdominal pain.

Each complication was divided into 19 groups. The groups were formed after all the journal was reviewed. Most of the complications were confirmed by endoscopy except for bronchopneumonia, which was detected by radiography (x-ray). Fever, purpura hemorrhagica and bastard stangles were based largely on clinical examination. Horses euthanized were calssified into those due to economic versus poor prognosis. If samples were taken but no results were found in the journal these horses was excluded from in this study of which one horse was thus excluded.

Owners of those horses for which telephone numbers could be found in the system were contacted by the author to detect the long term outcome for horses after their visit at the hospitals and to asses the long term outcome. Anamnesis questions were asked about the horse’s condition how the horse’s condition developed after time and if any other problems appeared. The owners were informed about the project and its aim. It was not possible to have

prepared several questions since the owners should not be lead to any answers. Of those owners which telephone contact was unsuccessful, email contacts provided this long term follow up for two of the cases.
The cases were sorted in excel and each clinical sign was given a code. The same was done with the complications. Each code was plotted in excel. The four most frequently observed clinical signs and complications were assessed for correlation by the McNemar´s test with significance set at $p<0,05$ using one-tailed method since there was great clinical variation between journals.
Litterature search was done in PubMed, Web of Knowledge and in text-books. The search words were strangles, *Streptococcus equi* subsp. *equi*, horse, equine, complications, bastard stranges and metastatic strangles, purpura hemorraghica and myositis. The review was used to find other articles on the topic.

LITERATURE REVIEW

Etiology

Strangles has been described in early veterinary science literature. Jordanus Ruffus reported it the first time in 1251. The name *Strangles* was coined because of the enlarged lymph nodes that made affected horses suffocate by obstructed airways (Sweeney et al., 2005).
Strangles is caused by *Streptococcus equi* subsp. *Equi* (hereafter called *S. equi)*, a grampositive bacterium in irregulary shaped cocci that forms long chain. It belongs to Lancefield´s group C and makes ”honey-colored”, mucoid colonies on blood agar. It also makes a wide zone of hemolysis on blood agar (see figure 1). The bacterium is encapsulated and highly virulent. There is a less virulent, atypical variant that produces a ”matte” appearance on blood agar. This one in also less encapsuled (Taylor and Wilson, 2006).
S. equi is highly host-adapted and causes disease only in horses, mules and donkeys (Timony, 1993).

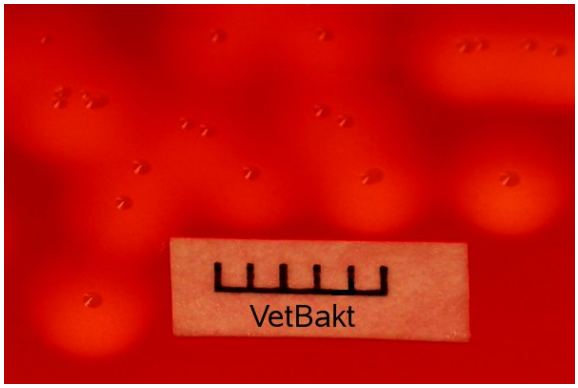

Figure 1 (vetbact.org)

Pathogenesis

Streptococcus equi enters via the mouth or nose (Sweeney et al., 2006).

In a study performed by Timoney and Kumar, University of Kentucky USA (2008), unexposed horses and ponies were inoculated with *S. equi* and followed the progression using serial postmortems. The study showed both nasal- and oropharyngeal tonsils were entery portals. The substrate of stratified squamous epithelium is special for the oropharyngeal tonsils. The epithelium of oropharyngeal tonsils is several layers thick. This thickness is unlikely to allow a rapid route of transition. It is more likely to be a temporary stop for the bacteria. The changes of internal pressure during inhalation pulled the bacteria into the underlying lymphoid tissue. *S. equi* were observed in tonsillar crypts within a few hours of inoculation. Cryptal entry may also be the explanation of penetration of the nasopharyngal tonsil. A few hours after intranasal inoculation the heavy growth of the bacteria cultured from tonsil surfaces contrasts with the very small number seen beneath the surface. This suggests that very few bacteria were involved in the initial phase of penetration. The bacterium has been detected in the lymph nodes as soon as 3 hours post-infection. Horses and ponies euthanized after onset of fever had tonsils heavily infiltrated with neutophils, both in the tonsillar crypt and through the tonsillar epithelium. Long chains of the bacteria were visible in both lymph nodes and tonsillar epithelium.

The neutrophils and the large numbers of extracellular *S. equi* are the explanation of release of complement chemotactic factors. It is generated by the interaction of C3 with bacterial peptidoglycan. The inability of the neutrophils to phagocytose the bacteria appears to be due to the combination of the hyaluronic acid capsule, antiphagocytic M-protein and a leucocidal toxin which all are released by the bacteria (Mukhtar and Timoney, 1998).

Other antiphagocytic factors are eg. fibronectin-binding factors, fibrinogen-bindning factors and bacterial enzymes (Harrington, Sutcliffe and Chanter, 2002). Meehan, Lynagh, Woods and Owen showed in their paper from 2001 for the first time that fibrinogen-binding protein (FgBP) binds to IgG and fibrinogen. This demonstrated that FgBP plays an important role in resistance to phagocytosis.

The extracellular bacteria accumulate in a form of long chains surrounded by large numbers of degenerating neutrophils. Streptolysin S and Streptokinase may also lead to the development of abscesses and lysis of damaging cell membranes (Sweeny et al., 2006). It is also Streptolysin S that is responsible for the beta-hemolysis on blood agar plates (Flanagan et al., 1998).

Clinical signs

The incubation period is 3-14 days after exposure (SVA, 2013).

The first clinical signs are acute onset of fever > 39°C and upper respiratory catarrh. The fever and depression are the only symptoms in 24-48 hours before the onset of nasal discharge proceed (SVA, 2013).

Younger horses are more likely to develop more severe symptoms than older horses, with acute swelling and abscess formation in retropharyngal and submandibular lymph nodes that subsequently rupture and drain. Older horses develop a mild form characterized by nasal discharge, smaller abscess formation and rapid recovery. The severity of disease also varies

depending on the immune status of the horse. Acute swelling and abcess formation in the lymph nodes lead to pharyngitis. A common sign of pharyngitis is dysphagia and the affected horses become anorexic and often stand with the neck extended. Nasal reflux of water and food can follow. Depression with fever and anorexia is a common sign as well as rhinitis and nasal discharge. Nasal discharge is initialy serous and rapidly becomes mucopurulent and later purulent, tenacious and profuse, often bilateral. Apprroximately one week after infection the affected lymph nodes become swollen and painful but the first sign is often painful, hot, diffuse edema. Serum can ooze for several days as the abcess mature before rupturing. The pus is creame coloured but does not have a foul odor. Although retropharyngeeal and submandibular lymph nodes are more affected other lymph nodes such as parotid and cranial cervical lymph nodes are also frequently involved. Parotidal abscesses can cause swelling of the eyelids. Some horses develpop a soft, moist cough but coughing is not a significant sign in many cases allthough squeezing the larynx may cause pain and extended neck position.

The guttural pouches

Horses have developed extensions from their auditory tubes called the guttural pouches. They are normaly just filled with air and divided into two parts, one lateral and one medial with the styloid bone between them. The two pouches have no connection with each other, but are only separated by a thin layer of connective tissue. The function of the guttural pouches is not yet defined but cooling the cerebral blood supply appears to be one. On the floor in the compartments run several important anatomical structures such as cranial nerves (IX-XII), the internal carotid artery and the sympatic nerve trunk (Dyce et al., 2002).

Retropharyngal lymph nodes may drain into the guttural pouch and cause empyema. Both retropharyngal abcsesses and gutteral pouch edema may cause swelling over Viborg´s triangle and may cause respiratory distress. The purulent material may become inspissated and form chondroid masses. Some affected horses will require emergency tracheostomy as a life-saving proceedure. In one study 4 of 15 horses had upper respiratory tract obstruction, which required tracheostomy, and 2 of these died of the obstruction (Sweeny, Whitlock and Meirs, 1987).

Diagnosis

The diagnosis of strangles in based on clinical symptoms and laboratory findings including culture, PCR and/or serology. However culture of nasal swabs, nasal washes and aspirated pus from abscesses remains gold standard. The bacteria is plated on agar with 5 % sheep or horse blood and incubated at 37°C. *S.equi* is differentiated from *S.zooepidemicus* using the aspect that *S.zooepidemicus* capacity to ferment sorbitol and lactose. *S. equi* is not present on the mucosa until 24-48 h after onset of fever. Thus isolation of horses with fever to limit transmission is advocated (Sweeney et al., 2005).
Nasal washes are more effective than swabs in detection of small numbers of bacteria. The washes can take sample from a greater surface area. The technique is although simple. The first thing to do is to wipe the external part of nasal cavity with saline. 120 ml temperated saline is flushed into the nasal cavity by a 50 cm long soft rubber tube (12 fench, 5-6 mm

diameter). The tube is directed in to the cavity towards the eye. The saline is collected in a sterile rectal glove and sent to the lab in a sterile Falcon tube (milk tube) (sva, 2013).
PCR, polymerase chain reaction, is a method to detect DNA sequence of SeM. SeM is the gene for the antiphagocytic M-protein of *S.equi*. The gene is also found in *S.zooepidemicus* but there is no evidence that the M-protein is expressed by that bacteria (Sweeney et al., 2005). PCR does not distinguish between live and dead organisms. PCR is approximately 3 times more sensitive than culture (Newton et al. 2000).
It is possible to detect DNA of SeM from *S.equi* in guttural pouch lavage. PCR is also useful to detect asymptomatic carriers and ascertain the success of elimination from the guttural pouch (Sweeney et al. 2005).
A study from Båverud, Johansson and Aspan (2007) showed that real-time PCR for differentiation and detection of *S.equi* was found to be reliable. And another study from Lindahl et al. (2013) found that real-time PCR in a combination with culture could detect over 90% of horses with clinical signs of strangles in one single sampling.
According to SVA serology is not used in Sweden. However serological testing is used abroad, particularly when material for PCR or culture testing cannot be obtained. Levels of SeM antibody in serum can be measured with an ELISA. It can be helpful diagnosing recent infection or determine the need for a booster vaccine. Serum titers peak 4 to 5 weeks after infection and remain high for 6 to 8 months (Sheoran et al., 1997).

Hematology and other diagnosis

Hematologic changes are leucocytosis, a high-segmented neutrophil count and high fibrinogen (Timoney, 1993).
Other diagnostic tools that may be required include radiography of the pharyngeal region, guttural pouch endoscopy and lymph node ultrasonography (Taylor and Wilson, 2006).

Vaccination

There are 3 commercially avaliable vaccines in the U.S. None of these provide complete protection, but may decrease the severity and incidence of the disease. No vaccine is registated in Sweden. The side effects are variable and thus vaccination should only be performed in susceptible horses at high risk of exposure or on endemic premises (Taylor and Wilson, 2006).
The side effects are injection site reaction, nasal discharge, lymphadenitis, submandibular abscesses and occasionally even purpura haemorrhagica. The side effects are both seen in IM-injection as well as IN (intra nasal) vaccination (Waller and Jolley, 2007).

Epidemiology

Strangles is said to be the most frequently diagnosed infectious disease of horses worldwide (Waller, Paillot and Timoney, 2011).
Transmission of *S. equi* occurs either direct or indirect from nasal discharges or lymph nodes discharges from affected horses. Direct transmission may be horse-to-horse contact with mutral head contact as normal equine behavior. Indirect contact occurs when sharing housing,

water sources, tack and twisters, feeding devices and clothing (Reed, Bayly and Sellon 2010). Transmission by buckets was early recognized by Sollysol in 1664 (Timoney, 1993).

Survival of *S. equi* in the environment is not particulary long. Experiments described in Veterinary clinics of North America by Timoney (1993) indicate that *S.equi* suspensions placed on wood or glass (which was sterilized) surfaces at 2 °C and 20 °C survived for 7-9 weeks. However these colonies were left at constant humidity and temperature and did not have any other microbials to compete with. This suggested that the natural survival time is less than experimental. Nonetheless there is a lack of field-based proof for environmental persistence.

S.equi is not part of the normal nasopharyngal bacterial flora. Most infected horses begin shedding two to three days after the onset of fever. Some animals never shed. New cases can therefore be isolated before they transmit infection but already have fever (Sweeney et al., 2005).

ACVIM by Sweeney et al. (2005) suggested that there is evidence that some horses may continue shedding the bacteria for several weeks after clinical signs have disappeared. However in the majority of horses *S.equi* is no longer detectable four to six weeks after total recovery. Based on the The concensus statement of the ACVIM a recovered horse may be a potential source of infection for at least 6 weeks after total recovery with no clinical signs.

Newton et al. (1997) reported that 1% to 10% of infected horses fail to clear the bacteria within four weeks. Shedding in the absence of clinical signs was documented for up to 39 months after recovery.

Horses of all ages can be infected but the disease is most common and severe in younger horses (Timoney, 1993). The infection is most common in horses 1 to 5 years of age (Reed, Bayly and Sellon, 2010).

S.equi is thought to infect only Equidae. However fatal pneumonia caused by *S.equi* was reported in an Ethiopian dromedary camel in 1997. The clinical signs were similar to those in strangles but postmortem examination showed fatal transformations in the lung (Yigezu et al., 1997).

A case of human strangles that caused bacteremia and meningitis in a horse handler was reported in 1986. The clinical signs were fever and acute facial swelling. Human infection is nevertheless very rare (Breiman and Silvetblatt, 1986).

Immunity

Immunity is good following natural infection in most horses. Studies referred by Taylor and Wilson (2006) suggested that approximately 75 % of the horses are protected for at least 4 years. But 25 % of the infected horses fail to develope an appropriate immune response and are susceptible to reinfection within 6 to 12 months.

Foals up to 3 months of age born from immune mares are resistant to strangles. A study made by Galan et al. (1986) observed IgA and IgG antibodies in sera and nasal secretions of foals after colostrum ingestion. Titera were similar in mare sera, foal sera and colostrum samples.

But the immunity is unfortunately not lifelong. Epidemiologic studies specified in Reed, Bayly and Sellon (2010) report attack rates (the risk of getting strangles again) in horses greater than 3 years of age of 18%, 29% and 35%.

Detection of carriers

Transmission of *S.equi* has been the focus of investigation during several years. But the source of transmission is likely outwardly healthy animals incubating the disease or inapparent carriers of the bacteria. They carry the bacteria in their upper respiratory tract. The purulent material in the guttural pouch thickens and form chondroid masses and is the best-recognized site of carriage the bacteria. Guttural pouch empyema develops and may be considered as a complication of strangles.

Chondroids can occur in very large numbers (Sellon and Long, 2007). These are unlikely to drain from the guttural pouches by themselves. Horses that are totaly recovered but continue to be infectious through periodic shedding are referred to as long-term, subclinical carriers. Introduction of these animals into new herds may be the reason for new outbreaks.

Diagnosis of the infection is best achieved by using endoscopy. Culture and PCR is best for detection by using lavage samples. Empyema and/or chondroids may also be diagnosed by radiography of the guttural area. However changes may not be visible in all cases (Sweeney et al. 2005) The chondorids may enable *S.equi* to persist in the guttural pouches and transmit to naive horses (Waller, Paillot and Timoney, 2011).

In a study by Newton et al. (2000), 14 asymptomatic carriers of *S.equi* were identified of which 13 horses showed evidence of carriage in the guttural pouch.

The review by Waller, Paillot and Timoney reports incomplete drainage of the pus in the guttural pouches in up to 10 % of the cases. Intermittent shedding from the guttural pouches occurs in normal horses as well as asymptomatic carriers (Waller, Paillot and Timoney, 2011).

Of 91 horses identified in a retrospective study by Judy et al. (1999) guttural pouch empyema was detected in 19 horses. In 14 of them *Streptococcus equi* was isolated.

Treatment of carriers

Appropriate treatment depends on the consistency and volume of the material. But repeated lavages using isotonic saline, with a lowering of the head together with an endoscope and eventually a suction pump is one. Administration of topical benzylpenicillin has been used to supplement the treatment. In a study by Newton et al (2000) one method of delivering a mix with gelatin and penicillin was reported. The mixture is said to be more effective in remaining present in the pouches than an aqueous solution. Topical treatment with acetylcysteine has also been used as a treatment (Sweeney et al., 2005).

An endoscopically guided memory-helical polyp retrieval basket may be used to remove the chondroids. Lavage may need to be repeated several times until they become normal and is confirmed as *S.equi* negative by result and/or PCR (Taylor and Wilson, 2006).

There are also some surgical treatement available. One is hyovertebrotomy and ventral drainage through Viborg´s triangle. Access to the guttural pouch may also be attained with laser surgery to create drainage through the healthy side and use of an endoscopic snare or basket to remove chondroids (Auer & Stick, 2012).

Treatment

There are markedly divided veterinary opinions whether or not to use antibiotic treatment. This includes even Sweden. In the beginning of this summer, 2013, the Swedish Veterinary Association published a consensus paper on antibiotic treatment i several conditions including strangles. Their opinion is that antibiotics can be used in the early stages of strangles in a combination with isolation (svf.se)

Sweeney et al. (2005) reported in their consensus statement that the majority of strangles horses do not need any treatment other than a proper rest in a dry and warm stable. Food and water of good quality should be easily accessible.

S.equi is sensitive to penicillin, chloramphenicol, erytromycin, tetracyclines and lincomycin. However procaine penicillin is the antibiotic of choice (Timoney, 1993).

If antibiotics are used they should be as an immediate therapy of new cases in the early acute phase with only fever and depression. This means the first 24-48 hour before nasal discharge proceed. Infected ponies treated with antibiotics and isolation at onset of fever did not develop lymph node abscessation in a study. When abscesses have not yet developed the antibiotics have sufficient access to the bacteria (Sweeney et al. 2005). According to Sweeney et al. (2005) antibiotics should be given for 3-5 days.

A case report by C.A. Piché (1984), from an outbreak with 479 horses showed that foals treated with intramuscular benzathine penicillin developed strangles after the treatment was discontinued. This suggested that protective immunity would not be stimulated when antibiotics are used early in clinical disease.

For horses that have developed lymph node abscessation are antibiotic therapy is probably contraindicated. Treatment only prolongs the enlargement and eventual rupture of lymph node abscesses. The infection and abscessation will return when the treatment is discontinued (Sweeney et al,. 2005). Therapy should instead be to enhance the maturation of the abscess and later drainage. Hot packs can be applied to speed up the maturation. However data reported in ACVIM consensus statement note that these techniques are doubtful. Surgical drainage of lymph nodes is sometimes neccesary. But if the drainage is made before the abscess has maturated enough it may only lead to minimal drainage of exsudate and continued lymph node swelling. Daily flushing may help to keep the abscess open.

NSAID medications may reduce fever, pain and the swelling and inflammatory reaction. This may improve eating and drinking.

How to manage an outbreak of strangles

The best way to prevent an outbreak of strangles is to be proactive. It is nessesary to have an isolation stable as quarantine. The horse should be isolated for 3 weeks (see incubation time) (Sellon and Long, 2007). Timoney suggested in his paper in Veterinary clinics of North America that rectal temperature should be taken twice daily.

It is of advantage to keep younger horses separated from older horses, where the most important thing is to keep horses in competition away from younger horses. The stable should be supplied with good equipment for hand washing with clean warm water, soap and a clean

towel. If for example several use the horse trailer it should therefore be cleaned after each use (sva.se, 2013).
Strangles has a very contagious nature, which makes the control of spread among horses on farms very difficult. Horses with strangles as well as their environment should be isolated. Owners and caretakers must be very careful not to spread the bacteria to other horses. In an ideal world separate clothing and shoes should be used (Taylor and Wilson, 2006).
Rectal temperature should be taken at least once daily. If a horse with fever is discovered it should be isolated immediatly. To find horses that are infectious after clinical recovery 3 sequential nasal swabs at weekle intervals should be taken (Sweeney et al. 2005).
Paddocks used by infected horses should be considered contaminated for 1 month thereafter (Timoney, 1993).
In Sweden, strangles is a notifiable disease (SJVFS 2013:23) .

Complications

Pneumonia

According to ACVIM suppurative bronchopneumonia is one important complication to strangles. In the clinical report from Sweeny et al. (1987) three of the six euthanized horses (out of 15 suffering from complications) had pneumonia. One horse developed upper respiratory tract obstruction. Of the 35 cases of complications reported by Ford and Lokai (1980) did 62 % (22 horses) died of pneumonia secondary to strangles.

Bastard stangles – metastatic spread

Bastard strangles is used to describe metastatic spread of the abscesses. The term was first appeared in European veterinary literature in the late 17th century. During this period the term ”Bastard” was used to describe atypical forms of diseases in both veterinary and human medicine. The use of the terme ”Bastard strangles” has been used to describe all kinds of strangles forms as ”retropharyngal lymph node abscesses draining to the guttural pouch” (Slater, 2003).
The metastatic spread of *S.equi* can occur in several ways. The most common routes are hematogenous and lymphatic. Other routes may be via connecting tissues or structures such as cranial nerves transport the bacteria or when the horse aspirates purulent material (Sweeney et al. 2005).
Common sites of infection are the spleen, liver, mesentery, lung, kindeys and brain.
Horses with bastard strangles often have symptoms such as intermittent colic and pyrexia, anorexia and weight loss and depression; with signs dependent on organ and site of localisation (Reed, Bayly and Sellon, 2010).
There is no consensus among veterinarians whether antibiotic treatment predisposes formation of internal abscesses. Ramey (2007) reports in his paper about this question from an evidenced point of view. Reasons why there might be more metastatic spread with antibiotic treatment are e.g. that antibiotics alter bacterial protein synthesis and therefore reduce immunogen levels or that antibiotics works on the bacterial wall and prevent immunity to develope. However Ramey clarified that there are no data supporting this association.

The diagnosis of metastatic spread strangles includes rectal examination, abdominocentesis and ultrasonography. CT can be performed in foals or small ponnies and nuclear scintigraphy may be used to determine the location of the abscesses. Internal abcesses may be difficult to differentiate from abdominal neoplasms on abdominal fluid analysis if no neoplastic cells are idintified, since both samples contain leukocytosis and hyperproteinemia. Hematologic and serum findings include anemia, neutrophilic and monocytic leukocytosis, hypoalbuminemia and hyperglobulinemia and hypocalcemia (Reed, Bayly and Sellon, 2010).

Ford and Lokai (1980) described in their case report from 1980 the prevalence of complications. In their study the mortality rate was 10 %. All were necropsied and of 35 dead horses, 5 suffered from gastrointestinal problems.

Sweeny et al. (1987) collected data from an outbreak in 1983 including 235 horses. All horses were vaccinated and 74 horses suffered from strangles. Their data suggested that 20,3 % (15 horses) of the horses developed complications and 2 of these 15 had mesenteric lymphnode abscessation. It is not known if any of the horses developed bastard strangles in later years.

Pusterla, Whitcomb and Wilson (2007) published a collection of medical records from University of California with 10 horses suffering from internal abdominal abscesses. All ten had classical symptoms and earlier described diagnoses were performed. The survival rate in this study was 40 %. Treatments for these horses were long-term treatment with antibiotics (mean duration 72 days), NSAIDs and intravenous crystalloid fluids.

Treatment of bastard strangles is difficult. Many horses in above mentioned studies were euthanaized due to their symptoms. But a case report from Berlin et al. (2013) described 4 successful medical treatments. These horses were treated with penicillin, NSAID and fluids and improved within days of treatement. The median duration of antibiotical treatment was 35 days (from 32 to 50 days).

Metastatic spread and manifestation may also occur in the brain, but is likely rare. 4 cases with neurological symptoms were decribed by Slater (2003). In 2 cases MRI (Magnetic resonance imaging) was used for confirmation, and in one case MRI was compared with CT. Due to severe symptoms and poor prognosis one of these horses survived (Spoormakers et al. 2003). The clinical signs depend on the location of the abscess. When the cerebrum is affected circling, blindness, depression and changed behaviour may occur (Reed, Bayly and Sellon, 2010).

Purpura hemorrhagica

Purpura hemorrhagica is an aseptic necrotizing vasculitis. It is a non-contagious disease and it appears to occur after reexposure to strangles in natural infection or vaccination.

The vasculitis is probably caused by an immune complex deposition in the blood vessels. This immune complex reaction is a type III hypersentitivity that develops when there is an excessive combination of antibodies with antigen that activate complement. When these complexes are disposited in the blood vessels the activated complement generates chemotactic peptides that attract neutrophils (Tizard, 2009)

Galan and Timoney (1985) characterized the immune complex in sera and showed that immune complex containing IgA and *Streptococcus equi*. A study made by Heath et al. (1991) showed an association between development of purpura hemorrhagica and IgA-

antibodies. IgA titers were higher in horses with the disease than horses that were not reexposed to *S.equi.*

The clinical sign develop within two to four weeks of an infection. The most common clinical signs are subcutaneous edema of all four limbs with exudation of serum, reluctance to move, haemorrhages on mucous membranes, epistaxis, fever, depression, anorexia, tachycardia, tachypnea and colic as a result of haemorrhage and necrosis of the intestinal wall. Haematological findings are neutrophilia, anemia, hyperptoteinemia, hyperfibrinogenemia, hyperglobulinemia and high levels of CK and ASAT (muscle enzymes). (Pusterla et al. 2003). Renal dynfunction in an case has also been described by Robers and Kelly (1982). The clinical signs vary from a mild to a severe and fatal form. Death may occur as a result of renal failure, cardiac arrhythmias, pneumonia, colic or severe muscle infarctions. The diagnosis is often based on clinical signs and anamnesis but may be confimed by skin biopsis showing leucocytoclastic vasculitis. (Reed, Bayly and Sellon, 2010).

As said, purpura hemorrhagica is a very severe disease. The study by Sweeney et al. (1987) reported that 4 out of 15 horses with complications had purpura hemorrhagica and one of the four was euthanized. In another study made by in 2003 reported 53 cases of purpura hemorrhagica. 22 of the horses were either infected or vaccinated with *S.equi* and three of them did not survive (Pusterla et al. 2003)

The primary treatment for the disease is corticosteroids (dexamethasone or prednisolone). When infectious is suspected antibiotics should be given. In the study by Pusterla et al. (2003) all 53 horses were given corticosteroids and 42 of them also received NSAID, flunixin or phenylbutazone. The dose was gradually reduced while the horses recovered. 26 horses had a suspected infection and were treated with antibiotics of different types. Supportive care as hydrotherapy, bandages with protective dressings and mild excercise was also provided. 23 horses were treated for more than 21 days.

Myositis

Muscle Infarctions

According to Sweeney et al. (2005) this complication is most likely a syndrome from purpura hemorrhagica. As mentioned aboved mildly elevated levels of muscle enzymes are detectable and titers of SeM-specific antibody may be increased. The clinical signs are muscle stiffness and lameness. Aggressive treatment is, with corticosteroids and antibiotics (penicillin and gentamicin). The condition Henoch-Schonlein purpura is a complication to streptococcal infections in humans, also due to immune complex. The clinical signs are also similar to those seen in horses (Duquesnoy, 1991).

Valberg et al. (1996) found in their study a relationship between *S.equi* infection and myopathies. Out of 17 horses with purpura hemorrhagica seven horses were detected with high CK and ASAT activities and postmortem examination confirmed muscle infarctions.

Rhabdomyolysis with Progressive Atrophy

This form of myositis has been reported in Quarter horses exposed to *S. equi.* A study by Sponseller et al. (2005) reported the connection with horses with *S. equi* infection. Some of these horses had underlying problems with a clinical history of polysaccharide storage

myopathy. Two of the horses had clinical history with *S. equi* infection. Muscle enzymes were increased.
A case report from Quist et al. (2011) described a Quarter horse that developed stiff, stilted gait and ventral edema. The gelding also showed signs of tachycardia and tachypnea. When examing the guttural pouches empyema consistent with strangles was found.

Agalactia

In the study by Sweeny et al. (1987) one broodmare with strangles developed agalactia. The agalactia is believed to be secondary to fever and anorexia rather than an infection in the mammary glands themselves. Nursing foals at their side may be requiring milk supplements.

Other complications

Other complications are myocarditis and glomerulonephritis there *Streptococcus equi* may be a trigger for development. Of 35 horses with complications in the study by Ford and Lokai (1980), 4 horses suffered from cardiovascular complications and 2 horses suffered from urogenital diseases.
Uveitis is also mentioned as a complication of strangles. In a study by Roberts (1971), 13 horses developed chorioretinitis 3 months after *S. equi* infection. The horses were shipped together to a ranch in California and once there developed typical clinical signs of strangles.

Most of the data on disease complications due to stranges is based on selected limited outbreaks and field observations. This present study examined wether the clinical picture in horses in Sweden that are referred to equine hospitals from a number of different outbreaks had a similar array of presenting clinical signs and what types of complications and long term prognosis was for the referred horses.

RESULTS

This retrospective study contained 69 horses referred to the equine hospitals in Uppsala and Helsingborg. All of the horses had a diagnosis of strangles in therin medical records.
The average age of the horses were 9 years with a range from 3 months to 26 years.
25 of the horses were mares, five horses were stallions and 39 were geldings.

Horses at referral hospital

Clinical signs (table 1) and complications (table 2) were recorded and the percentages were calculated.

Table 1

CLINICAL SIGNS	**n (horses)**	**PERCENT OF HORSES**
Fever	24	35 %
Dysphagia	22	31 %
Nasal discharge	14	20 %

Dyspnea and respiratory distress	13	19 %
Swollen lymph nodes	9	13 %
Abscess	7	10 %
No clinical signs	6	9 %
Coughing	4	6 %
Ataxia	4	6 %
Inappetence	4	6 %
Edema	3	4 %
Emaciation	2	3 %
Diarrhea	2	3 %
Colic	1	1 %
Cyanotic or other circulatory effects	1	1 %

Table 2

COMPLICATION	**n (horses)**	**PRECENT OF HORSES**
Swollen retropharyngeal lymph nodes	24	35 %
Purulent discharge in the guttural pouch	21	30 %
Chondroids	14	20 %
DDSP/ Swollen pharynx	12	17 %
Follicular hyperplasia	5	7 %
No endoscopic findings	5	7 %
Abscess parotid region	4	6 %
Abscess submadibular lymph nodes	4	6 %
Mucosal changes in the guttural pouch	4	6 %
Cranial nerves impact	3	4 %
Euthanized (economic reasons)	3	4 %
Euthanized (poor prognosis)	2	3 %
Purpura hemorrhagica (not confirmed diagnosis)	2	3 %
Positive test results	2	3 %
Negative test results	1	1 %
Bronchopneumonia	1	1 %
Fever	1	1 %
Bastard strangles (not confirmed diagnosis)	1	1 %
Died by natural causes	1	1 %

Correlation clinical signs and complication

A McNemar´s test was made for comparison of the relationship of the four most common clinical signs to complications (table 4-5). The aim was to see if any clinical sign eas associated with one or more complication.

Table 4

Clinical sign		**Swollen LN (a)**		**Purulent in guttural pouch (b)**	
Fever	Pos	Pos	11	Pos	9
	Pos	Neg	13	Neg	15
	Neg	Positive	13	Pos	12
	Neg	Negative	32	Neg	33
Dysphagia	Pos	Pos	10	Pos	7
	Pos	Neg	12	Neg	15
	Neg	Pos	14	Pos	14
	Neg	Neg	33	Neg	33
Dyspnea	Pos	Pos	7	Pos	4
p(a) < 0,05	Pos	Neg	6	Neg	9
	Neg	Pos	17	Pos	17
	Neg	Neg	39	Neg	39
Nasal discharge	Pos	Pos	5	Pos	5
p(a) < 0,05	Pos	Neg	9	Neg	9
	Neg	Pos	19	Pos	16
	Neg	Neg	36	Neg	39

Table 5

Clinical sign		**Chondroids (c)**		**DDSP/Swollen pharynx (d)**	
Fever	Pos	Pos	11	Pos	6
p(d) < 0,05	Pos	Neg	13	Neg	18
	Neg	Positive	13	Pos	6
	Neg	Negative	32	Neg	39
Dysphagia	Pos	Pos	6	Pos	6
p(d) < 0,05	Pos	Neg	15	Neg	16
	Neg	Pos	14	Pos	8
	Neg	Neg	33	Neg	39

Dyspnea	Pos	Pos	2	Pos	3
	Pos	Neg	11	Neg	10
	Neg	Pos	14	Pos	9
	Neg	Neg	44	Neg	47
Nasal discharge	Pos	Pos	4	Pos	3
	Pos	Neg	10	Neg	11
	Neg	Pos	10	Pos	9
	Neg	Neg	45		46

Intrestingly, there are a lack off association between nasal discharge and chondroids and purulent discharge in the guttural pouches.
Purulent discharge in the guttural pouch and guttural pouch chondroids were the second and third most common complications. It is clinically interesting to see how many horses that had both chondroids and purulent discharge, since both are detectable only largely by endoscopy. It is valuable to see whether chondroids might be found together with purulent discharge (table 6), however surprisingly there was no correlation between these two complications ($p>0,05$).

Table 6

Chondroids		**Purulent discharge guttural pouch**	
p= 0,140521	Pos	Pos	2
	Pos	Neg	12
	Neg	Pos	19
	Neg	Neg	36

Horses euthanized

Out of the 69 horses five were euthanized and one died of natural causes at the hospital (table 7). Three of them had dysphagia, three nasal discharge and two dyspnea or respiratory distress as clinical signs. The most common complication in these horses was chondroids. But one should remember that chondroids also was the third most common complication among all horses. The one horse that died by natural causes had two common clinical signs, dyspnea and nasal discharge, but also cyanotic mucous membranes. The horse was a 10-month-old foal and a post-mortem exam was not done. The dyspnea was severe and the foal was suddenly found dead in the morning.
The author did not see any correlation between clinical signs and euthanasia. Except from the horse with impact on the circulation. The only detectable correlation was between chondroids and euthanasia. All three euthanized horses had chondroids.

Table 7

	Euthanized or died by natural causes at hospital		
Horse	**Clinical signs**	**Complication**	**Reason for euthination**
1	Dyaphagia + Swollen LN	Chondriods + Purulent discharge guttural pouch	Economic

2	Nasal discharge	Chonroids	Economic
3	Nasal discharge	Chondroids	Economic
4	Dysphagi + Dyspné	Chondroids + Swollen retropharyngeal lymph nodes + Swollen pharynx	Animal welfare
5	Dyspnea+Nasal discharge+Cyanotic membranes	Swollen retropharyngeal lymph nodes	Died by natural causes
6	Dysphagia + Emaciation	Abscess parotid region + Abscess submandibular lymph nodes	Died at home, no information

Follow up

Out of the 69 horses, 51 horses were contacted by telephone. 19 of the telephone numbers were disconnected. 27/32 horses were completely resolved and one horse was sold directly after being sent home. Four horses were euthanized (table 8). Three horses were euthanized as a consequence of strangles. The forth horse was euthanized due to being diagnosed as a wobbler.

Table 8

	Euthanized at home		
Horse	**Clinical sign**	**Complications**	
1	Fever + Abscess	No information	Animal welfare
2	Fever + Abscess + Swollen lymph nodes	Purulent discharge guttural pouch	Economic
3	Dysphagia+ Swollen lymph nodes	Swollen pharynx + Abscess parotid region	Animal welfare
4	Other		Other reason

DISCUSSION

The aim of this retrospective study was to determine the nature of the presenting clinical signs and diagnosis of complications in horses admitted to two referral equine hospitals in Sweden, for the diagnosis Strangles, and to evaluate whether specific clinical signs were related to complication rates or to long term outcome.

There are many studies and reviews made on this subject during the last decades. Strangles is a highly contagious disease and may cause many problems due to both isolation and animal welfare.

By contacting the owners the author could find out if any complications after infection had occured. The aim was to find out if there were any complications not written in the journal. A difficulty in this study was to find these horses after an infection. Four horses were euthanized, three of them due to complications to strangles. The horse euthanized by economic reasons had surgery recommended for removal of purulent discharge in guttural pouch.

Sweeney et al. published in 2005 a consensus statement with guidelines for strangles. This consensus statement (ACVIM) is one of the most widely known reports of complications to strangles today. But it is important to remember that this is *Streptococcus equi* infection according to American circumstances. In the consensus statement by ACVIM pneumonia is ”an important sequel of strangles”. Two of the most highly cited articles are Ford and Lokai (1980) and Sweeney et al. (1987).

The article by Ford and Lokai (1980) is a case report from a yearling farm with an outbreak of strangles. It that study 62 % of the horses that died had pneumonia as a complication to strangles.

In the study by Sweeney et al. (1987) 26 % (4 out of 15) developed pneumonia. All four were euthanized. In that article pneumonia secondary to strangles was a frequent cause of death and they suggest this as consistent with earlier reports by Ford and Lokai (1980).

The author’s opinion is that these studies are highly cited. The study by Ford and Lokai is highly cited as evidence that strangles is associated with a high rate of pneumonia. The author´s opinion is that this reported pneumonia as a complication to strangles without consideration of other reasons. Those horses came to the farm from many different farms. Pneumonia can be caused by other reasons such as transport related pleuropnemonia. It is remarkable that the study by Ford and Lokai is so highly cited to when strangles was not the most probable reason for most of the mortality mentioned in the study. The study by Sweeney et al. also mentions pneumonia and while it is the author´s view is that strangles may have been a primary disease it is noteworthy that other pathogens and reasons could have caused the pneumonia secondary to strangles.

In this study one horse, a 3 moths old foal, developed bronchopneumonia. X-rays confirmed the diagnosis. The foal had respiratory distress, swollen lymph nodes and an abscess in the inguinal region. He was treated with penicillin and NSAID and was unfortunately lost to follow up. An abscess in the inguinal region is not the primary location of strangles, but *S. equi* was found in this abscess. The author’s opinion is that while bronchopneumonia may be a sequel of strangles many other pathogens may also cause pneumonia in foals, but that were not ruled out. None of the other horses in this follow up developed pneumonia or any other respiratory disease except reinfection with strangles.

The author´s conclusion is that the complication rates of 62 % and 26 % suffering from pneumonia as reported by Ford and Lokai (1980) and Sweeney et al (1987) are far higher than those found in the current study.

The most common presenting clinical signs seen in this study were fever and dysphagia. Separating dysphagia from inappetence and emaciation was important. Dysphagia included problems with swallowing, chewing and food intake. Emaciation included horses with none

of these clinical signs but weight loss. Of the horses survived with dysphagia as a clinical sign, 31 % survived. That is higher than mentioned in other publications.
Not many horses (13 %) suffered from swollen lymph nodes which is mentioned to be one of the most common clinical signs. If a detailed clinical exam was performed those should clearly have been detected. A problem might be that the veterinarians only record it in the journal if the lymph nodes are markedly enlarged.
Dyspnea and respiratory distress and nasal discharge were both found in 19-20 % of the horses. Tracheotomy was required in one horse that had permanent displacement of the soft palate.
Ataxia was found in four horses but the relationship to clinical strangles is unclear. One of the horses had swollen retropharyngeal lymph nodes, one horse had chondroids and the remaining horses had no endoscopic findings or other signs of complication.

Purpura hemorrhagica is a severe complication from strangles. In a study above mentioned by Sweeney et al. (1987) 27 % of the horses suffering from complications developed purpura hemorrhagica. According to ACVIM concensus statement the risk of developing purpura hemorrhagica from vaccination is not known. But since there is no licensed vaccine available in Sweden this was not an issue in this paper.
Purpura hemorrhagica was found in 2/69 (3 %) of the horses; none of them with confirmed diagnosis, yet having typical clinical signs. Those horses arrived to the hospital with edema (both ventral and distal). One horse had the edema only the first day and it resolved on its own. One horse received corticosteroids and recovered and another horse recovered despite not being administered with corticosteroids.

Bastard strangles is likely the most well known complication to strangles. In the study by Ford and Lokai (1980) the mortality rate was 10 % in horses suffering from bastard strangles. Whereas Sweeney et al. (1987) had a mortality rate of 13 %. A similar mortality rate was not found in this study. However, one should keep in mind that the limited mumbers of horses in this study. It is important to remember that the author might have missed some horses with colic symptoms in the follow up. Both studies by Ford and Lokai and Sweeney et al. were referring to strangles in field cases. Nonetheless cases in this report were referred to hospital and should have received better treatment and monitoring and thereby increase likelihood of detection of bastard strangles should it have occured.
In this study was one horse suspected of developing bastard strangles. The clinical signs were emaciation, fever and colic signs in a 10 years old horse. The horse was euthanized but no post mortem exam was performed. The horse was suspected of having bastard strangles since the horse developed fever and colic signs for which bastard strangles would be a highly likely diagnosis. It is difficult to find horses with bastard strangles since these symptoms may be considerd as colic without connection to strangles.

Myositis is another complication mentioned in the literature. ACVIM consensus statement by Sweeney et al (2005) suggested that this is a complication to purpura hemorrhagica. This study did not find any horses with myositis. One reason could be that myositis associated to strangles without purpura hemorrhagica is uncommon and that not many horses in this study

developed purpura hemorrhagica. The generic form of myositis such as Rhabdomyolysis with Progressive Atrophy is uncommon in Sweden and it is presumably therefore no horse was found with that. Another reason may be that clinicans have misinterpreted muscoskeletal signs or that diagnosis of myositis was based on muscle enzyme elevations, which are not routinely monitored in strangles cases in Sweden.

Agalactia was not found in this study. Only one mare with its foal came to the hospital with clinical signs, which included nasal discharge but no fever.

The most common complication in this study was swollen retropharyngeal lymph nodes (33 %) which was detected by means of endoscopy. Out of those 23 horses were seven treated with lavage of the guttural pouches and two horses recieved local bensylpenicillin. All horses were treated with penicillin and NSAID.
Swollen retropharyngeal lymph nodes were correlated with dyspnea and nasal discharge and thus these latter clinical signs are indications for endoscopic examination of the guttural pouches.
The second most common complication (30 %) was purulent discharge in the guttural pouch, in all cases secondary to swollen lymph nodes that had ruptured. Seven horses of these 21 horses were treated with lavage of the guttural pouches, three of those with acetylcysteine, one with corticosteroids and one with local benzylpenicillin. Since treatment regimen differs between veterinarians this was not possible to analyze further.
Chondroids were the third most common complication (20 %) and had no correlation with purulent discharge in guttural pouch or any clinical sign. Thus they can be clinically silent and only detected by accident, yet serve as a source for infection to immunologically naïve horses.
Swollen pharynx was correlated to fever and dysphagia. The correlation to dysphagia is not suprising since the swollen pharynx may be presumed to cause difficulties in swallowing and chewing.
There was an surprising lack of correlation between nasal discharge, chondroids and purulent discharge in the guttural pouches. The same interesting lack of association was seen between dyspnea and swollen pharynx/DDSP.
The follow up was possible in 32/69 horses. Three horses were euthanized due to reinfection with strangles. No horse developed any other complication.

The conclusion from this work is that complications to strangles are less common than mentioned in earlier studies and reviews from other countries and other horse population settings. In particular, the reported high rate of pneumonia as a common complication to stangles was not observed in these Swedish conditions. As well, Bastard strangles, purpura hemorrhagica and myositis are not as common in Sweden as mentioned in earlier highly cited studies. No horse in this study suffered from endocadritis, glomerulinephritis or agalactia.

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